

Product Information

Matrix Metalloproteinase-7 human

recombinant, expressed in *E. coli*

Product Number **M4565**

Storage Temperature -70°C

EC 3.4.24.23

Synonyms: Matrilysin 1; MMP-7; uterine MP; putative MP I (PUMP1)

Product Description

Human Matrix Metalloproteinase-7 (MMP-7) is a matrix metalloproteinase that has been substrate-affinity purified from recombinant human promatrilysin expressed in *E. coli*.

Matrix Metalloproteinase-7 (MMP-7) may be used as a control for immunoblotting and ELISA, as well as for enzyme kinetics assays and substrate assays. By SDS-PAGE, a band at ~ 20 kDa is detected.

The matrix metalloproteinases (MMPs) are a family of at least eighteen secreted and membrane-bound zinc-endopeptidases. Collectively, these enzymes can degrade all the components of the extracellular matrix, including fibrillar and non-fibrillar collagens, fibronectin, laminin, and basement membrane glycoproteins. In general, a signal peptide, a propeptide, and a catalytic domain containing the highly conserved zinc-binding site characterize the structure of the MMPs. In addition, fibronectin-like repeats, a hinge region, and a C-terminal hemopexin-like domain allow categorization of MMPs into the collagenase, gelatinase, stomelysin, and membrane-type MMP subfamilies.¹⁻³ MMPs contain the motif His-Glu-X-X-His (X represents any amino acid) that binds zinc in the catalytic site, as well as another zinc ion and two calcium ions structurally. They fall within the matrixin subfamily and are EC designated 3.4.24.x. This group also contains astacin, reprolysin, and serralyisin, as well as other more divergent metalloproteinases. All MMPs are synthesized as proenzymes, and most of them are secreted from the cells as proenzymes. Thus, the activation of these proenzymes is a critical step that leads to extracellular matrix breakdown.

MMPs are considered to play an important role in wound healing, apoptosis, bone elongation, embryo development, uterine involution, angiogenesis,⁴ and tissue remodeling, and in diseases such as multiple sclerosis,^{2,5} Alzheimer's,² malignant gliomas,² lupus, arthritis, periodontitis, glomerulonephritis, atherosclerosis, tissue ulceration, and in cancer cell invasion and metastasis.⁶ Numerous studies have shown that there is a close association between expression of various members of the MMP family by tumors and their proliferative and invasive behavior and metastatic potential.

The tissue inhibitors of metalloproteinases (TIMPs) are naturally occurring proteins that specifically inhibit matrix metalloproteinases and regulate extracellular matrix turnover and tissue remodeling by forming tightly bound inhibitory complexes with the MMPs. Thus, TIMPs maintain the balance between matrix destruction and formation. An imbalance between MMPs and the associated TIMPs may play a significant role in the invasive phenotype of malignant tumors. MMPs and TIMPs can be divided into two groups with respect to gene expression: the majority exhibit inducible expression and a small number are produced constitutively or are expressed at very low levels and are not inducible. Among agents that induce MMP and TIMP production are the inflammatory cytokines TNF- α and IL-1 β . A marked cell type specificity is a hallmark of both MMP and TIMP gene expression (i.e., a limited number of cell types can be induced to make these proteins).

Matrix Metalloproteinase-7 was first discovered in the involuting rat uterus. MMP-7 may have a role in processes such as host defense, cell proliferation, and protein turnover as well as tissue remodeling. MMP-7 is the smallest member of the matrix metalloproteinase family of endopeptidases and lacks the COOH-terminal hemopexin regulatory domain or hinge region shared by other MMPs. Like the other MMPs, MMP-7 is secreted as a zymogen, then activated. The 28 kDa zymogen is reduced to ~19 kDa (calculated) by enzymatic cleavage after the conserved cysteine switch.

MMP-7 is expressed in epithelial cells of normal and diseased tissues. It is not constitutively produced, but rather, its synthesis is induced in specific tissues. MMP-7 substrate specificity is broad, most closely resembling the activity of MMP-3. MMP-7 degrades collagens IV and X, gelatin, casein, laminin, aggrecan, entactin, elastin, versican, and fibrinogen. MMP-7 is activated by plasmin and MMP-3, and is also implicated in the activation of other proteinases such as plasminogen, MMP-1, MMP-2, and MMP-9. It is frequently expressed in various types of cancer including colon, stomach, prostate, and brain cancers. MMP-7 is over-expressed in 80% of human colorectal cancers and is known to be an important factor for early tumor growth, with potential function also in tumor progression, invasion, and metastasis. In addition, MMP-7 also regulates intestinal α -defensin activation in innate host defense,⁷ releases TNF- α in a model of herniated disc resorption, and cleaves FasL to generate a soluble form in a model of prostate involution.⁸ MMP-7 is up-regulated by the tumor promotor, phorbol 12-myristate 13-acetate (PMA), TNF- α , EGF, and IL-1.

The human MMP-7 gene has the chromosomal location of 11q21-q22.

Reagent

Supplied in a buffer containing the active enzyme in 10 mM HEPES, 0.15 M sodium chloride, 5 mM calcium chloride. No endogenous inhibitors, TIMPs, are detected in the recombinant product.

Concentration: ~0.1 mg/mL

One unit of enzyme activity is defined as the digestion of 1 μ g of azocoll per minute at pH 7.5 and 37 °C.

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

The product ships on dry ice and storage in aliquots at -70 °C is recommended. Repeated freezing and thawing is not recommended. Storage in frost-free freezers is not recommended.

References

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GA,PHC 01/17-1